Case Reports

Psittacosis

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HUMAN PSITTACOSIS is a respiratory or systemic illness caused by infection with *Chlamydia psittaci*. It is contracted by being exposed to discharges of infected birds; therefore, it is a zoonosis. The disease is called psittacosis if acquired from psittacine birds (parrots, parakeets and related species) and ornithosis if acquired from nonpsittacine birds (chickens, pigeons, pheasants, turkeys, ducks). It may rarely be contracted from nonavian sources such as cattle, sheep, cats or other humans. The disease has also been reported in laboratory personnel working with chlamydiae. If recognized early and treated appropriately with tetracycline, the disease generally has little morbidity and low mortality. Failure to consider the diagnosis, however, may result in prolonged illness, serious sequelae and a higher mortality.

Two recent cases of typical psittacosis underscore the importance of obtaining an epidemiologic history, more specifically, a history of exposure to birds or other vectors, in patients presenting with fever and pulmonary infiltration.

Reports of Cases

Case 1

The patient, a 35-year-old male lithographer, was admitted to hospital for treatment of left lower lobe pneumonia in May 1985. Nine months before admission, left pleuritic chest pain without fever developed and continued for three or four days. A chest roentgenogram showed a left pleural effusion, and there was a mild leukocytosis (11,700 per μl). Findings of thoracentesis were a yellow fluid, pH 8.1, a protein level of 5.2 grams per dl and glucose 96 mg per dl. The leukocyte results were lost. Levels of lactic dehydrogenase (LDH) from pleural fluid and serum specimens were 162 and 85 IU per liter, respectively. The patient was treated with a ten-day course of erythromycin, 250 mg four times a day, and sent home. A purified protein derivative (PPD) skin test (5 tuberculin units) was negative at 48 hours; a skin test for mumps was positive. Cultures of pleural fluid specimens for bacteria, fungus and acid-fast bacilli were negative, and cytologic studies showed only reactive mesothelial cells. Ten days later the patient felt well and results of a physical examination were entirely normal.

Six days before admission, a persistent fever to 40.5°C (104.9°F) developed, and he had delirium, shaking chills, generalized myalgias and malaise. Two days later he had a cough productive of scanty, thick yellow sputum, substernal chest pain, headache and anorexia. He had no photophobia, neck stiffness, adenopathy, rash, arthralgia, diarrhea or dysuria.

The next day he had a fever to 39.1°C (102.4°F) and coarse breath sounds at both bases. A 0.3-cm nodule in the left axilla was thought to be a skin abscess and was incised and drained. The leukocyte count was 9,500 per μl with a left shift, and a chest roentgenogram was normal. The examining physician thought he had a viral syndrome, prescribed acetaminophen and sent him home. Nausea, emesis and crampy abdominal pain occurred during the hours before hospital admission.

The patient was single, heterosexual and lived in a rooming house in San Francisco. He did not smoke cigarettes or use alcohol or intravenous drugs. Three months before his illness, he had killed and skinned two rabbits. His landlady's two pet cockatiels (apparently domestic) had died the same month (May) of unknown causes, and his cat had had a recent gastrointestinal illness.

On physical examination, he was acutely ill, in a mildly toxic condition and somewhat anxious. The temperature was 39.1°C, the pulse rate 120 beats per minute, respirations 12 per minute and unlabored and blood pressure 110/70 mm of mercury. There was a small, erythematous macule in the left axilla (at the site of prior incision and drainage). The pharynx was injected but without exudate. A small, tender left axillary node was palpable. The neck was supple, and there were coarse rales at the left posterior base with egophony and bronchial breath sounds. The heart had a tachycardic, regular rhythm and summation gallop, but no murmur. There was mild right upper quadrant tenderness, but no hepatomegaly, and one observer noted a palpable spleen tip. Findings of a neurologic examination were nonfocal.

The results of a complete blood count and serum chemistry tests were normal, except for a serum sodium level of 130 mEq per liter; the erythrocyte sedimentation rate was 50 mm per hour, and analysis of urine showed a specific gravity of 1.024, 2+ protein and 1+ ketones. A chest x-ray film showed a left lower lobe infiltrate and blunted left costophrenic angle consistent with a small pleural effusion (Figure 1). Gram's stain of a sputum specimen showed moderate polymorphonuclear leukocytes and scattered Gram-positive cocci. Cultures of sputum, blood and urine were negative for pathogens, and serum specimens were taken for serology. The rapid plasma reagin test for syphilis

(McPhee SJ, Erb B, Harrington W: Psittacosis. West J Med 1987 Jan; 146:91-96)

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ABBREVIATIONS USED IN TEXT

ALT = serum alanine aminotransferase
AST = serum aspartate aminotransferase
Ig = immunoglobulin
LDH = lactic dehydrogenase
P_{CO_2} = partial carbon dioxide pressure
P_{O_2} = partial oxygen pressure
PPD = purified protein derivative

was nonreactive, and the titer for tularensis agglutinins was less than 20. The patient was initially treated with tetracycline, 500 mg four times a day; erythromycin, 500 mg four times a day, was added when his temperature rose to 40.3°C (104.5°F).

His condition gradually improved, with the fever and cough resolving over the next several days. Following discharge, convalescent serum specimens were obtained. Acute and convalescent complement fixation antibody titers for *Chlamydia* confirmed the diagnosis of psittacosis, with a fourfold rise from 1:8 on May 21, 1985, to 1:32 on June 3, 1985. There was no change in complement fixation antibody titers for influenza A or B, adenovirus, *Mycoplasma* or Q fever. A month later he was entirely well.

Comment. This man had typical pneumonitis and systemic features of the acute form of psittacosis. The occurrence of acute pneumonitis and splenomegaly suggests the diagnosis. Although the culture-negative pleural effusion nine months before admission might have also been due to *C psittaci* (because antibodies are not protective), the initial complement fixation titer was probably too low for this possibility. The history of exposure to rabbits suggested the diagnosis of pleuropulmonary tularemia, but exposure occurred three months before the illness, making it unlikely. Although *C psittaci* infection may be acquired from cats (and the patient's cat had been ill), the patient's infection was most probably acquired from one of the two pet cockatiels that had been purchased recently.

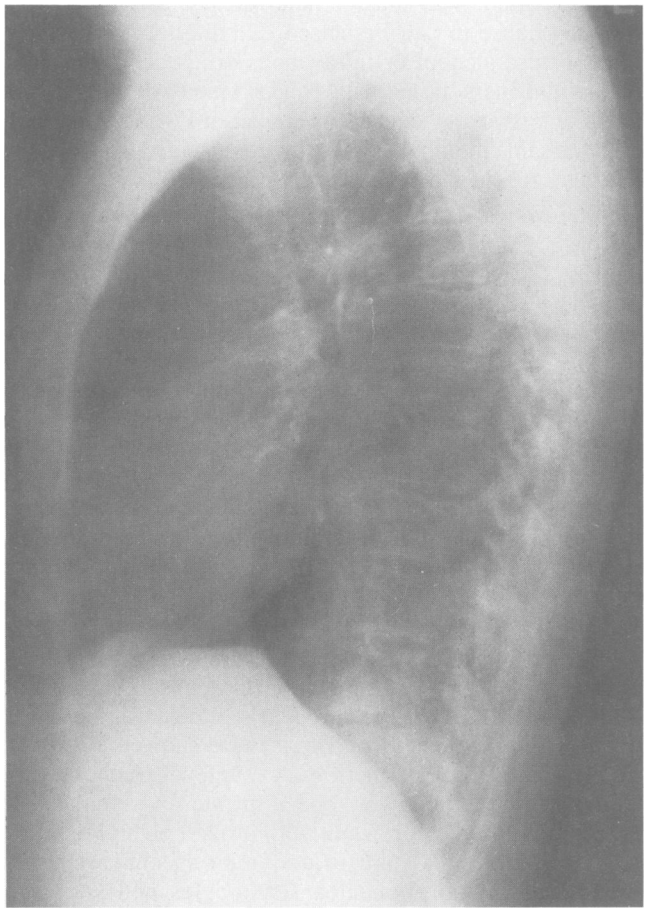

Figure 1.—A lateral chest radiograph of the patient in case 1 shows a consolidation of moderate size in the left lower lobe and a small left pleural effusion.

Case 2

The patient, a 35-year-old licensed vocational nurse, had been in excellent health until September 1982, when she noted fatigue and both her hands were cold. The next day she noted malaise and had a fever to 38.8°C (101.8°F), lightheadedness, nausea and mild right upper quadrant and midepigastric abdominal pain; the following day anorexia and mild frontal headache occurred. On the third day her temperature increased to 39.5°C (103.1°F) and rigors developed. On physical examination the following day, she had a temperature of 38.8°C, mild right upper quadrant tenderness and an epigastric bruit. Serum chemistry tests showed mild elevations of serum aspartate aminotransferase (AST, formerly SGOT) and serum alanine aminotransferase (ALT, formerly SGPT) levels, with a normal serum alkaline phosphatase value. A Monospot slide test for infectious mononucleosis was negative. She was told she probably had viral hepatitis and was sent home. Her fever was not suppressed by taking aspirin.

The following day, increasing malaise and a persistent, nonproductive cough developed. Her temperature ranged between 39.5°C and 40°C (104°F), and her physician noted rales in the left lower lung. A chest x-ray film showed right upper and lower lobe infiltrates. Results of a complete blood count were normal; the erythrocyte sedimentation rate was 85 mm per hour. Cold agglutinins were absent. Serum chemistries showed an AST level of 28 and an ALT value of 70 IU per liter. Arterial blood gas determinations made while the patient was breathing room air showed a pH of 7.51, a partial oxygen pressure (P_{O_2}) of 65 torr and a partial carbon dioxide pressure (P_{CO_2}) of 20 torr. Cultures of sputum, blood and urine specimens were negative for pathogens. Ultrasound examination of the abdomen was unrevealing. She was treated with nafcillin sodium and tobramycin given intravenously, but her condition did not improve. One week after the onset of symptoms, fever persisted and she had increasing dyspnea. Arterial blood gas measurements with the patient breathing room air showed pH 7.51, P_{CO_2} 20 torr and P_{O_2} 64 torr.

She had been previously well except for mild hypertension—treated only with dietary sodium restriction—and a history of hyperthyroidism with exophthalmos, treated with antithyroid medications. A PPD skin test had been positive one year previously; the chest roentgenogram had been normal and no therapy had been prescribed.

On physical examination on admission, she appeared moderately ill and had dyspnea. The temperature was 39.5°C, the pulse rate 130 per minute, the respirations 24

per minute and the blood pressure 105/75 mm of mercury. There were a few urticarial lesions on the thighs and knees. The conjunctivae were injected, the neck was supple and there was no adenopathy. Chest examination showed dullness and decreased breath sounds at both bases posteriorly, and there were coarse rales and bronchial breath sounds over the entire left and lower right lung fields posteriorly. There was a grade 2/6 systolic ejection murmur. There was mild right upper quadrant and epigastric tenderness; the hepatic span was 8 cm to percussion. The spleen was not palpable. An epigastric bruit was again noted. A stool specimen was positive by guaiac test. The remainder of the examination showed no abnormalities.

Results of a complete blood count, differential and serum chemistry tests were normal, except for elevated values for liver function tests (serum alkaline phosphatase 130 IU per liter, AST 46 IU per liter and LDH 182 IU per liter). A urinalysis showed 1+ protein, trace heme, 5 to 10 leukocytes and 5 to 10 erythrocytes per high-power field. A chest roentgenogram showed right upper and bilateral lower lobe infiltrates and bilateral small pleural effusions (Figure 2). Results of arterial blood gas analyses with the patient receiving five liters of oxygen per minute by nasal prongs showed pH 7.51, Po_2 76 torr and Pco_2 25 torr. Gram's stain and acid-fast smear of sputum specimens were negative for pathogens. Cold agglutinins were absent. Cultures of sputum, blood, urine, cervical and stool specimens were negative. A coccidioidomycosis counterimmunoelectrophoresis test was negative, and *Legionella* and *Mycoplasma* complement fixation antibody titers were done and subsequently were negative. The patient was initially treated with a regimen of nafcillin sodium, tobramycin and erythromycin.

The following day, it was learned that one month before admission the patient's husband had brought home a pet cockatiel from a private aviary in El Sobrante, California. About two weeks later, the bird had become ill with lethargy and rhinitis and was given chlortetracycline hydrochloride (Aureomycin) for five days. The patient became ill toward the end of the bird's antibiotic therapy.

Subsequent serologic studies showed the following microimmunofluorescence antibody titers: anti-*Chlamydia psittaci* immunoglobulin (Ig)G 1:128, anti-*C psittaci* IgM 1:128, anti-*Chlamydia trachomatis* IgG 1:32 and anti-*C trachomatis* IgM 1:32. The antichlamydial antibody complement fixation titer was 1:16.

The patient was thereafter treated with tetracycline, 500 mg four times a day. Her condition improved rapidly over the next several days, with resolution of the chest abnormalities and improvement of the arterial blood gas values to pH 7.46, Pco_2 33 torr and Po_2 80 torr with the patient breathing room air. She was discharged on the sixth hospital day and an additional 18-day regimen of tetracycline was prescribed. A similar respiratory illness had developed in the patient's 9-year-old daughter, who was also treated with a tetracycline regimen. Her cockatiel was given Keet Life (chlortetracycline) for 45 days. The State Department of Public Health was contacted and the aviary placed under quarantine until further investigations were conducted.

Comment. Both severe multilobar pneumonia with hypoxemia and mild nonspecific hepatitis from psittacosis developed in this woman. She became progressively ill despite treatment with broad-spectrum antibiotics until the history of bird exposure was elicited, the diagnosis of psittacosis considered and tetracycline therapy begun. Her daughter probably also had a mild case of psittacosis from

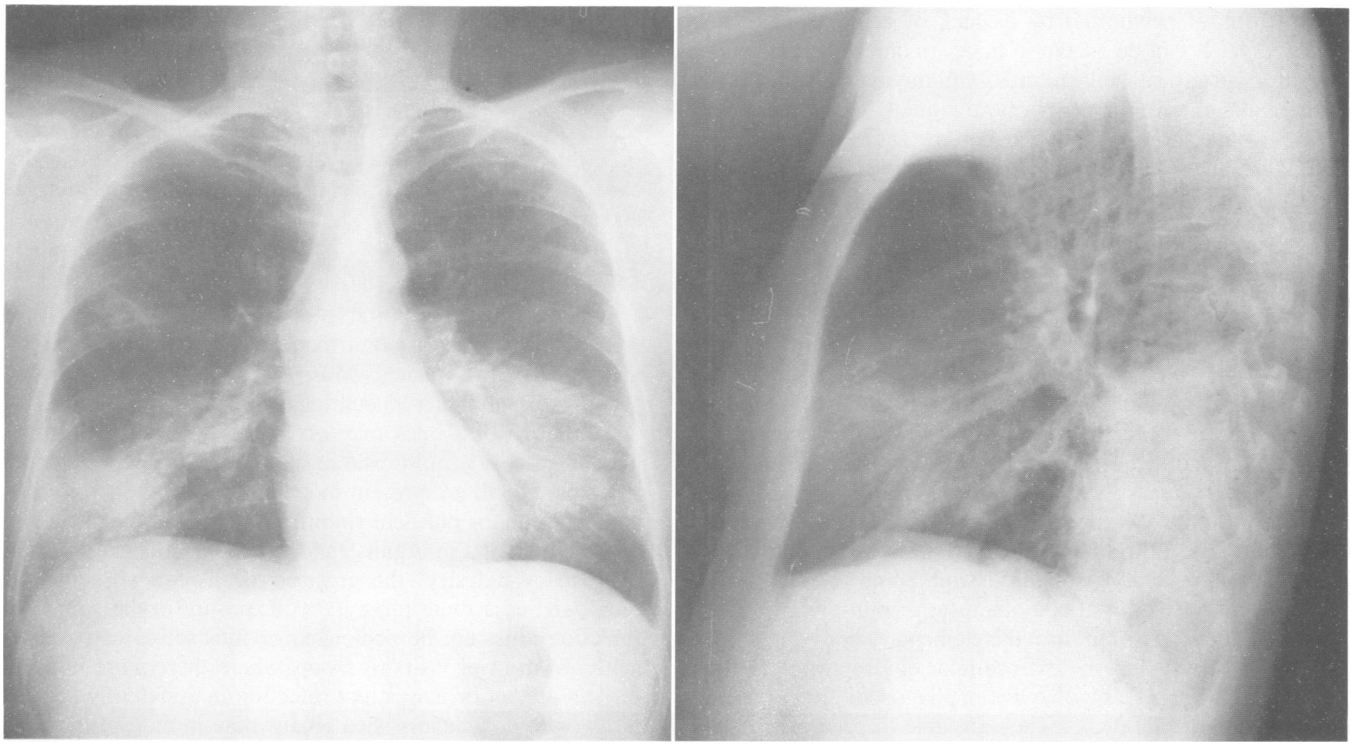

Figure 2.—Left, Posteroanterior and **right,** lateral chest radiographs from case 2, showing patchy consolidation in both lower lobes, focal nodular density in the right upper lobe and small bilateral pleural effusions.

exposure to the household source. Appropriate measures were taken to ensure eradication of the infection both in the patient's home and at the aviary.

History

The history of psittacosis is only a century old. Jurgensen may have been the first to describe the typical pneumonia of psittacosis. Ritter recognized the first outbreak of human psittacosis in 1879 in Ulster, Switzerland. Seven cases of fever, pneumonia and stupor followed in people exposed to sick birds, and three proved fatal.[1] Following 4 cases (2 fatal) in 1882 in Bern, Switzerland, and 47 cases (13 fatal) in Paris in 1892, Morange in 1895 coined the term "psittacosis" from the Greek *psittakos* (parrot). An epidemic, however, in 1929-1930 of 750 to 800 cases originating in Argentina and affecting the United States, Europe and Africa led to the discovery of the responsible agent. In 1930 Krumweide and Armstrong in the United States, Levinthal in Germany and Bedson in the United Kingdom discovered the responsible organism. Levinthal, Coles (United Kingdom) and Lillie (United States) described coccobacillary forms seen under light microscopy, subsequently known as Levinthal-Coles-Lillie bodies. Also in 1930, according to Schachter and Dawson, Bedson and Western devised the complement fixation antibody test that has remained the mainstay of diagnosis.[2]

Microbiology

Chlamydia psittaci (responsible for psittacosis) can be readily distinguished from *Chlamydia trachomatis* (differing serotypes of which are responsible for lymphogranuloma venereum, trachoma, inclusion conjunctivitis, nongonococcal urethritis, cervicitis, salpingitis, proctitis, epididymitis and pneumonitis of newborns) by its lack of cell-wall staining with iodine. It can be grown in mice, in chick egg embryos and in a variety of cell cultures. Chlamydiae are obligate intracellular microorganisms, more similar to bacteria than viruses in that they are sensitive to some antibiotics, contain both DNA and RNA and have discrete cell walls, resembling those of Gram-negative bacteria. Because they cannot synthesize adenosine triphosphate and require other metabolites for growth, they must live within cells. When they infect the host cell, they take over its synthetic processes and direct them toward producing more chlamydiae.[3-5]

The developmental cycle of chlamydiae is complex, lasting about 48 hours. The 350-nm elementary body is the infectious particle, capable of attaching to the surface of susceptible host cells, such as epithelial cells of the respiratory tract, and of inducing its own phagocytosis. Once inside the cell, the elementary body reorganizes within six to eight hours to form the 800-nm metabolically active initial (reticulate) body. The initial body then synthesizes new materials and divides repeatedly by binary fission. Some 18 to 24 hours after infection, the initial body then stops multiplying, reorganizes and condenses to form the elementary body, which is capable of survival in the extracellular environment. In the cell environment, chlamydiae live in phagosomes throughout their developmental cycle. They are able to prevent phagosomes from fusing with lysozomes until very late in the cycle.[3-5]

Clinical Manifestations

Psittacosis is rare in children younger than 10 years; it is more severe in adults older than age 40.[6] The incubation period varies from 7 to 15 to as much as 30 to 39 days, the shorter incubation periods resulting from heavier exposure.[2]

Presentations vary considerably with a predominance of respiratory tract symptoms (nonproductive cough, dyspnea, pleurisy, epistaxis, sore throat and, rarely, hemoptysis) and systemic symptoms (fever, malaise, rigors, sweats, headache, anorexia, nausea and vomiting and abdominal pain). Less common are photophobia, conjunctival suffusion, generalized arthralgias, myalgias, stomach cramps and diarrhea. Physical signs include fever, rales, pulmonary consolidation, pharyngeal erythema, hepatomegaly, altered mental state, pleural friction rub, slow pulse rate relative to temperature and splenomegaly.[6] Rales or rhonchi are detected in about a fifth of patients, but the majority have roentgenographic evidence of pneumonia. Relative bradycardia is noted in about a third of patients.[7-12]

Unusual manifestations of psittacosis include erythema nodosum, pericarditis and pericardial effusion, myocarditis and congestive heart failure, hepatic granulomata, myositis, rhabdomyolysis, reactive arthritis, encephalitis, abortion and hemolytic anemia.[13-25] In addition, there have been several recent reports of culture-negative infective endocarditis caused by *C psittaci*. The clinical presentation has generally been one of subacute endocarditis of the mitral or aortic valve, some cases failing to respond to prolonged tetracycline therapy and requiring valve replacement.[26-32]

Pathology

In birds the primary sites of disease are the liver, spleen and pericardium; in humans the lung is most often involved.[5,6] The pulmonary lesion is a lobular interstitial pneumonitis, extending from hilum to periphery, typically of dependent lobes and segments. There may also be severe tracheobronchitis. The spleen shows gross and microscopic findings of acute splenic tumor; the liver, nonspecific hepatitis; the heart, interstitial myocarditis, edema and subendocardial hemorrhage, and the brain, lymphocytic meningitis and arachnoiditis.

Epidemiology

Psittacosis is a reportable disease. In 1979 there were reported 116 cases of confirmed or presumptive psittacosis and 1 death in 25 states. Most cases were reported in the West, with California accounting for 27 (23%).[33]

Although bird bites may occasionally result in disease, humans usually acquire psittacosis by inhaling *C psittaci* organisms, which are present in exudates of birds with serous conjunctivitis or purulent rhinitis, in dried excrement in the environment and in organs and feathers of infected birds. In the poultry industry, the greatest risk is for workers who pluck and eviscerate turkeys.[9,11] Exposure to the source of infection must not be prolonged or intense; cases have resulted from brief visits to rooms where there were infected birds.[10] Latent or inapparent infection in apparently healthy birds (who nonetheless shed chlamydiae in their excrement) may represent low levels of multiplication held in check by host defenses.[5] In addition, human-to-human transmission

and transmission by other animals, including cattle, sheep and cats may rarely occur.[34,35] A carrier state for *C psittaci* was reported by Meyer and Eddie in 1951.[36]

Differential Diagnosis

The differential diagnosis of psittacosis is that of atypical pneumonia. It includes both relatively common illnesses, such as *Mycoplasma pneumoniae,* Legionnaire's disease and the viral pneumonias (influenza, parainfluenza, adenovirus, varicella, cytomegalovirus and respiratory syncytial virus) and uncommon causes of atypical pneumonia, such as Q fever, pleuropulmonary tularemia and *Pneumocystis carinii* pneumonia. Tuberculosis, atypical mycobacterial infection and fungal infection (especially histoplasmosis and coccidioidomycosis) must also be considered.

The best clues to the correct diagnosis of psittacosis are a history of exposure to birds in a person with a flulike illness or a chest roentgenogram that shows pulmonary infiltrates far more extensive than the symptoms or physical findings would suggest.[12]

Diagnosis

All chlamydiae share the same complement-fixing antigen. Thus, the widely used complement fixation test does not distinguish between *C psittaci* and *C trachomatis.* The clinical syndromes caused by each, however, are sufficiently different for this to be a minor problem.

Antibodies usually appear in the second to fourth week of illness, but early and prolonged treatment with tetracycline may delay, decrease or suppress antibody production, making serologic diagnosis difficult. Diagnosis may be aided by comparing complement fixation antibody titers early in the illness, throughout the convalescence and several months later; alternatively, a physician may be aided by obtaining blood or sputum specimens for isolation of chlamydiae before beginning antibiotic treatment.[37]

The complement fixation test is useful in diagnosis if appropriately timed (acute and convalescent) serum specimens show rising antibody titers. Seroconversion is defined as an increase of fourfold or greater, or a titer of less than 1:8 rising to more than 1:16. If a specimen is not obtained during the acute phase, single titers of 1:64 to 1:256 or more support the diagnosis. Although isolation techniques are no longer particularly difficult, the risk of infection to laboratory workers is great enough to advise that it be attempted only in specially equipped laboratories.[2]

The Centers for Disease Control define a confirmed case as a clinical specimen positive for *C psittaci* or an illness characterized by any combination of fever, chills, lower or upper respiratory tract disease, myalgias, headache, photophobia and splenomegaly plus a fourfold or greater rise in complement fixation antibody titer to 1:32 or more on two serum specimens obtained two or more weeks apart and studied in the same laboratory. A presumptive case is defined as a compatible illness and either an antibody titer of 1:32 or more on a single specimen or a stable titer of 1:32 or greater on two or more serum specimens.[33]

Newer diagnostic tests that distinguish between the two species, such as enzyme-linked immunosorbent assays and monoclonal antibody techniques, are now being evaluated.[38]

Morbidity and Mortality

Host defenses against chlamydiae may involve both cell-mediated and T-cell-dependent humoral immune responses. Humans show no prolonged immunity, and relapses or reinfections have been reported even for persons with high complement fixation antibody titers.[7]

Before antibiotics, the untreated disease usually lasted two to three weeks, but fever and illness sometimes persisted for two months or longer. Convalescence was prolonged and relapses occurred.

Mortality was 10% to 40% in the preantibiotic era.[10] Today, estimated mortality ranges from 1% to 5% (but, if the disease is recognized early and treated appropriately, mortality can be as low as 0.5%).[9] About a fourth of patients in recent epidemics have required hospital admission for 4 to 16 days.[11]

Prevention and Treatment

Chlortetracycline-impregnated birdseed and quarantine programs for imported birds have been used in an attempt to reduce the spread of the disease.[2] A study by Schachter and colleagues, however, shows that, despite such measures, the reservoir of infection persists. During a one-year period, they tested tissue specimens from 101 ill or dying parakeets and parrots in the United States for *C psittaci*; 37 (37%) were positive.[39]

Experimental efforts to immunize humans against psittacosis have been disappointing.[3]

The treatment of choice for psittacosis is tetracycline, 2 to 3 grams given in divided doses daily. Erythromycin is suggested as an alternative in children and pregnant women or in adults allergic to tetracycline, but may not be as effective. Experience with other antibiotics (chloramphenicol and rifampin) is limited.[2,10] To prevent relapses, therapy with tetracycline should be prolonged (anecdotal evidence suggests three weeks).

Defervescence after therapy is instituted may occur within one to six days.[12] A dramatic response to tetracycline, with the temperature dropping from 40.6°C (105.0°F) to normal within four hours has been reported.[40] In some cases, however, defervescence may be quite slow. The use of acetaminophen should probably be avoided because it may accentuate the hepatitis produced by psittacosis; such a reaction may prove fatal.[41]

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Nine Episodes of Infective Endocarditis in One Patient—A New Record

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(Simonson J, Schaaf VM, Mills J: Nine episodes of infective endocarditis in one patient—A new record. West J Med 1987 Jan; 146:96-98)

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INFECTIVE ENDOCARDITIS is a frequent complication of intravenous drug abuse, and recurrence is common if parenteral drug abuse continues.[1-3] Right-sided endocarditis in drug abusers has a mortality rate of 5% to 15%, while the mortality for each episode of left-sided endocarditis is 24% to 64%.[3-5] The likelihood of surviving several episodes of left-sided bacterial endocarditis is therefore small, and previous publications have reported no more than six episodes in a single patient.[6] We report the case of a patient who has survived nine documented episodes of mitral valve infective endocarditis over a 12-year period, including two episodes of fungal endocarditis and two mitral valve replacements.

Report of a Case

The patient is a 34-year-old man with a history of intravenous drug abuse since 1967. His first episode of mitral valve endocarditis was diagnosed in October 1972, when he came to San Francisco General Hospital Medical Center (SFGHMC) with fever and a murmur of mitral regurgitation. Multiple cultures of blood specimens grew group D *Streptococcus*. The patient was treated with a regimen of penicillin G and streptomycin sulfate given intravenously for 30 days and recovered without complication. He continued to use intravenous drugs and had eight subsequent episodes of endocarditis over the next 12 years (Table 1), all treated at SFGHMC. At least four of these episodes were complicated by congestive heart failure. Two episodes (numbers 5 and 6) were caused by persistent *Candida* infection complicated by bacterial superinfection. There was never any evidence of right-sided valve infection. In 1978 he underwent mitral valve replacement for persistent fungal endocarditis, and the two subsequent bacterial infections were cured medically, despite this prosthesis. In July 1984 he was diagnosed as having combined bacterial and fungal endocarditis and was given another mitral valve replacement (despite objections from some consultants) in conjunction with antimicrobial therapy. During the operation he was found to have abscesses in the mitral annulus. At last follow-up (November 1986), the patient was mildly disabled and receiving medical therapy for New York Heart Association class II congestive heart failure. He had no signs of infection and said he had discontinued intravenous drug use. Blood specimens drawn in November 1986 and cultured for bacterial and fungal pathogens were sterile.

Discussion

Recurrent infective endocarditis has been defined as a new intracardiac infection developing either more than six months after cure of an initial infection, regardless of pathogen, or by infection with a new organism regardless of time elapsed.[3,7] The remarkable case of the patient reported here met the first criterion in episodes 3 and 7, the second in episodes 4 and 5 and both criteria in episodes 2, 6, 8 and 9 (Table 1).

Recurrences of infective endocarditis are common in parenteral drug abusers. One study found that second episodes occurred in 17% to 24% of patients, and 5% had third episodes within a mean follow-up time of 20 months.[5] These rates are much higher than the 4% to 8% reinfection rate seen in the nonaddict population.[7] In 1979 Mokotoff and co-